The exact manner in which this operation gives relief in glaucoma is a matter of some doubt. Jonnesco[2] of Bucharest thinks that glaucoma is caused by a central lesion and that removal of the ganglion destroys the communication between this centre and the eye. The vaso-constrictor fibres are destroyed, blood-pressure is lowered, and extravasation is reduced. The excito-secretory fibres to the uveal tract are destroyed and the secretion of the aqueous is limited. The iris dilating fibres are destroyed, the pupil contracts, and the filtration angle is opened up. At the same time the fibres supplying the non-striped muscle in Tenon's capsule are cut off, so that the pressure on the efferent veins is removed.

From a consideration of this case it is clear that the contraction of the pupil, drawing the iris away from and so opening up the filtration angle following the removal of the ganglion, is a minor factor in the reduction of tension. It is obvious that the control of the sympathetic over the circulation and nutrition of the uveal tract is of much greater importance. We know that in many cases of serous cyclitis the filtration angle is free and unobstructed, and the rise of tension often seen is due to changes in the constitution of the intra-ocular fluid. It seems highly probable, therefore, from the rapidity with which the tension fell after the completion of the operation in the present case, although the pupil could not contract, that the good results which were seen after the performance of this operation were due, not so much to any change in the filtration angle brought about by contraction of the pupil, but rather to the abolition of the control of the sympathetic over the vascular apparatus and secreting epithelium of the uveal tract, whereby the character and amount of the intra-ocular fluid were materially altered.

Sympathectomy for glaucoma was first performed by Jonnesco in 1897 and since then has been carried out by many surgeons abroad and in England. Abadie,[3] Panas,[4] Ball[5] (St. Louis), Burghard,[6] Work Dodd,[7] and others have recorded cases. In Jonnesco's cases, which were chronic or subacute with only one acute case, very encouraging results were obtained. There was marked and permanent improvement in vision in all the chronic cases except those in which optic atrophy had set in, but there was no improvement in the acute case. In all cases the tension was immediately lowered and remained so and there was marked permanent contraction of the pupil with loss of pain in the eye. In the cases reported by Panas and Burghard the results of the operation were unsatisfactory and in Work Dodd's case disappointing. Ball's two cases were relieved, tension being reduced and vision improved, but there is no evidence that iridectomy would not have been equally effective. In Burghard's case intense pain at the side of the head and neck followed the operation. This unpleasant complication has been recorded by other operators. From a general consideration of the recorded cases, with the present large percentage of comparative and complete failures, it is probable that the severity of the operation as compared with iridectomy will prevent its performance in any but selected cases, particularly those in which iridectomy and sclerotomy have failed.

In glaucoma simplex the results have been more uniformly satisfactory than in inflammatory cases, but in the latter when iridectomy has failed sympathectomy holds out some hope of relief. In, however, most cases of glaucoma arising from obstruction in the filtration angle, especially acute cases, iridectomy will remain the operation of choice and will be found to give equally satisfactory, or even better, results than follow removal of the superior cervical ganglion of the sympathetic.

Leeds.

[2] La Presse Médicale, 1898.
[3] Archives d'Ophtalmologie, 1898.
[4] Ibid.
[5] Proceedings of the International Ophthalmological Congress, 1899.
[6] Brit. Med Jour., Oct. 20th, 1900, p. 1175.
[7] THE LANCET, Oct. 13th, 1900, p. 1071.

BRISTOL MEDICAL CHARITIES.—The late Mr. J. E. Exley bequeathed £1000 each to the Bristol Royal Infirmary, the Bristol Children's Hospital, and the Queen Victoria Convalescent Home, Bristol, also £500 to the Trowbridge Cottage Hospital. The residue of the property is left to his widow during her life in trust for the Bristol Royal Infirmary, which will eventually become entitled to about £15,000.

# ON THE PATHOLOGICAL CHANGES IN A CASE OF PROGRESSIVE MUSCULAR ATROPHY.

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DURING recent years many distinct clinical varieties of chronic muscular atrophy have been separated from progressive muscular atrophy. Hence cases to which the term "progressive muscular atrophy" can now be applied are rarely met with, and several writers have grouped together progressive muscular atrophy and amyotrophic lateral sclerosis as the same disease. In cases regarded as progressive muscular atrophy during life the lateral or crossed pyramidal tracts of the spinal cord have very often been found sclerosed more or less on pathological examination, even though there has been no spastic condition of the legs; and this is one reason for grouping together amyotrophic lateral sclerosis and progressive muscular atrophy as one pathological affection or one disease. A few rare cases are on record, however, in which during life the symptoms were those of progressive muscular atrophy (of the Aran-Duchenne type), and on pathological examination of the spinal cord the lateral or crossed pyramidal tracts were found to be normal. Such cases have been recorded by Dr. C. E. Beevor, Professor Strümpell, and MM. A. Dutil and J. B. Charcot and others. (In several cases there has been a slight degeneration of nerve fibres in the anterior ground bundle close to the anterior horns, though the crossed or lateral pyramidal tracts were normal; in other cases all parts of the anterior and lateral white matter were normal.) To the few cases hitherto recorded the following pathological report may be added; it appears to be worthy of publication owing to the rarity of such records.

The spinal cord of this case was kindly given to me by the late Dr. Leech of Manchester, who, had his life been spared, would probably have written the clinical portion of this article. My report will be devoted, therefore, to the microscopical examination; and to this I have added a brief clinical note of the case which I had the opportunity of examining from time to time, for many years, when the patient consulted Dr. Leech at the Manchester Royal Infirmary.

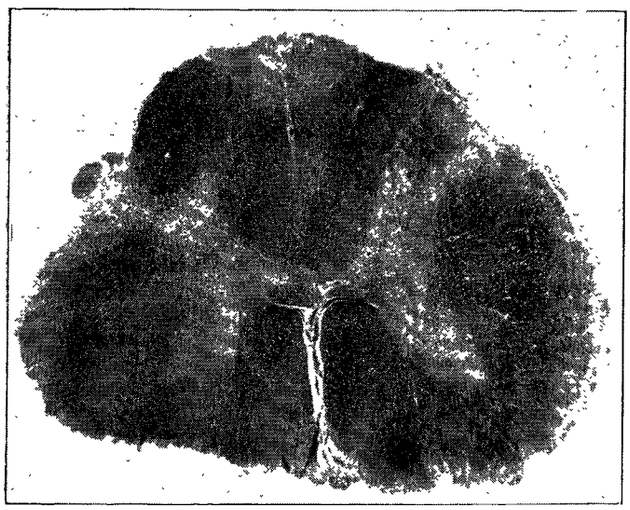
I first saw the patient, a man of middle age, in 1879. At that time both arms were paralysed, and the disease had commenced a few years previously. I last examined the man, along with Dr. Leech, in 1898. The patient died about 12 months later. The clinical features of the case were those of progressive muscular atrophy (of the Aran-Duchenne type) as described in the older text-books. There was slowly progressing muscular atrophy, with paralysis, affecting the small muscles of the hands, the forearm and the upper arm muscles, and finally the muscles of the neck. The arms for 20 years hung quite helpless by the side and the head was bent forwards. There was no affection of sensation, and the legs, bladder, and rectum were unaffected up to the time of the last examination. On many occasions I examined the condition of the legs with the object of ascertaining whether there was any evidence of spastic paralysis, but I was never able to detect any. When I last examined the patient in 1898, 12 months before his death and more than 20 years after the onset of the disease, the condition was briefly as follows. Both arms were markedly atrophied and completely paralysed and hung quite helpless by the sides. Every muscle of the hands and arms appeared to have atrophied completely. The head was bent forwards owing to the paralysis of the muscles of the neck. There were no symptoms of bulbar paralysis. The bladder and the rectum were unaffected. The patient was able to walk quite well. There were no indications of a spastic gait. There was no paresis of the muscles of the leg nor any rigidity to passive movements. The knee-jerks were present and there was no ankle-clonus. Sensation was not affected. Clinically, therefore, there were no symptoms to indicate any affection of the lateral pyramidal tracts of the spinal cord at the end of 20 years. I did not see the man during the last few months of his life and do not know the exact cause of his death. Dr. W. H. Pomfret, formerly surgical registrar at the Manchester Royal Infirmary,

having heard of the patient's death, succeeded in getting permission to make a limited post-mortem examination. He removed the spinal cord, the musculo-spiral nerve, and a piece of the adjacent muscular tissue of the forearm. To Dr. Pomfret, therefore, I am directly indebted for the specimens which he kindly forwarded to me with the permission of Dr. Leech. The specimens were hardened in Müller's fluid, embedded in celloidin, and the sections were stained according to Weigert's and Marchi's methods, and also with aniline blue-black and logwood. The nerve roots were stained according to Heller's pyrogallic and osmic acid method.

### MICROSCOPICAL EXAMINATION.

In the lower and middle cervical regions, on transverse section, whether stained with aniline blue-black, logwood, or according to Weigert's or Marchi's methods, the crossed or lateral pyramidal tracts of the white matter appeared to be quite normal (Fig. 1). Also the direct pyramidal tracts

FIG. 1.

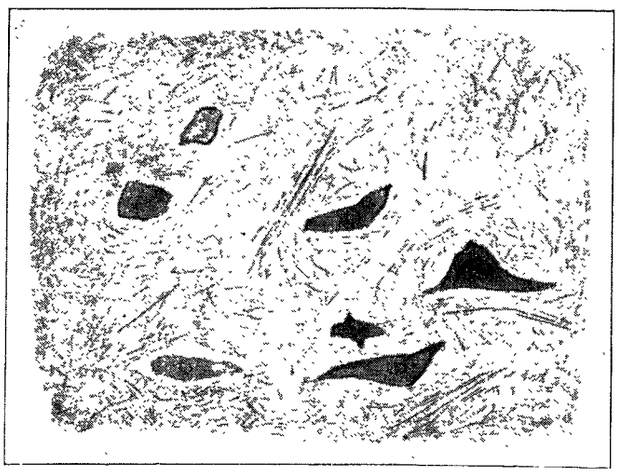


Transverse section of spinal cord, lower cervical region; progressive muscular atrophy; Weigert's stain; crossed pyramidal tracts normal. (Micro-photograph.)

and all the white matter of the anterior and lateral columns of the cord appeared to be normal with the exception of a very slight excess of neuroglia connective tissue in the region of the direct cerebellar tracts. The white matter in the antero-lateral columns close to the anterior horns appeared quite normal. In the anterior white commissure, in the sections stained according to Weigert's method, the nerve fibres were seen very distinctly. In the posterior median columns there was a very slight excess of neuroglia connective tissue (seen both in sections stained according to Weigert's method and in those stained with aniline blue-black), but the posterior external columns were normal. The anterior horns of grey matter were a little smaller than they are in sections of a normal cord at this region. The ganglionic nerve cells of both anterior horns had disappeared. After examining a number of sections of this region I was only able to find one atrophied nerve cell in the median region of one anterior horn. On examining sections stained according to Weigert's method the disappearance of nerve fibres in the anterior horns of the grey matter was very evident. The fine delicate nerve fibres (seen so clearly in the normal anterior horns in sections stained according to this method) were greatly diminished in number (Fig. 3). The fine nerve fibres of the posterior horns appeared to be normal. The nerve fibres from the posterior roots which enter the median side of the posterior horns were quite normal and were seen passing forwards to the intermediate grey matter, whilst the fine fibres in the surrounding median grey matter were diminished in number. In sections stained with logwood and eosin the nuclei of the neuroglia in the anterior horns were seen to be increased in number. The anterior nerve roots, even to the naked eye, appeared to be markedly atrophied; they were much smaller than the posterior roots and much less firm to the touch. On microscopical examination the posterior roots were normal, but the anterior nerve roots were seen to consist chiefly of fibrous tissue and empty nerve sheaths, with here and there a very few normal nerve fibres. The few remaining normal nerve fibres in the anterior roots were widely separated by fibrous tissue (as was well seen in specimens stained according to Heller's method). There was marked "hyaline" thickening of the walls of the small blood-vessels in the anterior median fissure and in the anterior horns of grey matter.

In the upper cervical region the condition was much the same. Most of the nerve cells in the anterior horns of grey matter had disappeared or atrophied markedly. The crossed or lateral pyramidal tracts and other parts of the

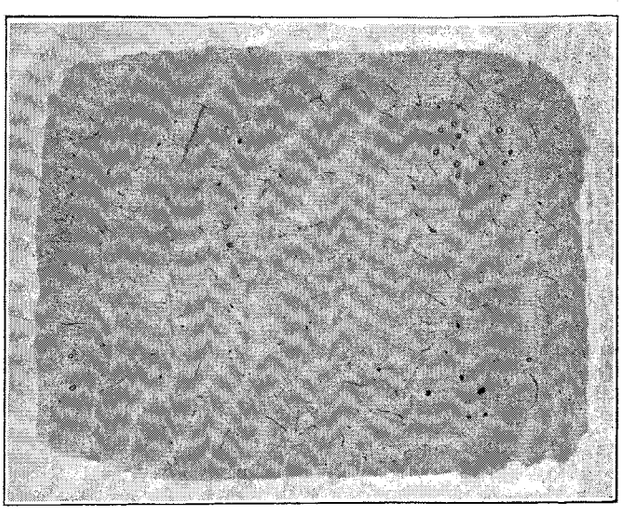
FIG. 2.



Normal anterior horn of grey matter, cervical region; Weigert's stain; showing nerve cells and numerous fine nerve fibres.

white matter were normal, with the exception of a very slight excess of neuroglia connective tissue in the posterior half of the posterior median columns. Sections of the dorsal region (upper and lower) presented normal appearances. The lateral or crossed pyramidal tracts, the posterior columns, and other parts of the white matter appeared to be normal both in sections stained according to Weigert's and Marchi's methods, as well as in those stained with aniline blue-black. Some of the nerve cells in the anterior horns and in Clarke's columns were pigmented, but otherwise the cells and fibres in both of these

FIG. 3.



Grey matter of anterior horn, lower cervical region; progressive muscular atrophy; Weigert's stain; nerve cells absent; fine nerve fibres very scanty.

regions appeared to be normal. The anterior nerve roots, to the naked eye and on microscopical examination, were normal in the dorsal region.

In transverse sections of the musculo-spiral nerve most of the bundles of nerve fibres appeared to be normal; in a few bundles there was a little increase in the connective tissue between the separate nerve fibres. The atrophied muscular tissue from the back of the forearm presented a very slight

resemblance to normal muscle to the naked eye, but it was very soft and flabby. On microscopical examination, however, the changes were most marked. Not a single normal striated muscle fibre could be detected. In place of the muscular tissue were collapsed muscle sheaths containing numerous elongated or round muscle nuclei which stained with logwood. Between these bundles of collapsed, well nucleated, muscle sheaths there was loose connective tissue, with a few clusters of large fat cells here and there. No evidence of hyaline or fatty degeneration of muscle fibres could now be detected.

The microscopical examination here recorded is of interest, because the case is one of the few which furnish clear pathological evidence that the crossed or lateral pyramidal tracts may appear normal in cases which have presented the symptoms of progressive muscular atrophy (of the Aran-Duchenne type). The crossed or lateral pyramidal tracts, from the highest cervical to the lowest dorsal region, presented no evidence of degeneration on microscopical examination, even in sections stained according to Marchi's and Weigert's methods. Also during life, up to the time of the last examination (more than 20 years after the onset of the disease) there were no symptoms of disease of the crossed pyramidal tracts.

Progressive muscular atrophy is closely allied to amyotrophic lateral sclerosis, and probably the pathological processes are in many respects similar; also there are transitional forms —cases corresponding clinically to progressive muscular atrophy, but pathologically to amyotrophic lateral sclerosis more or less. There is, however, a small group of cases which, both clinically and pathologically, differ from amyotrophic lateral sclerosis—cases such as that described in this article, and those previously recorded, to which I have referred. For these cases it appears advisable, at present, to retain the old name of "progressive muscular atrophy."

*References.*—C. E. Beevor: Article in Professor Clifford Allbutt's System of Medicine, vol. vii., p. 207 (London, 1899). J. B. Charcot: (Abstract) Neurologisches Centralblatt, 1896, p. 319. Déjerine: Comptes rendus de la Société de Biologie, 1895, p. 188. Dutil and J. B. Charcot: Comptes rendus de la Société de Biologie, July 28th, 1894. A. Strümpell: Text-book of Medicine, p. 654 (footnote), (London, 1893). Villers: (Abstract) Neurologisches Centralblatt, 1897, p. 842.

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# TETANY.

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TETANY, which is not an uncommon disease in children, is somewhat rare in adults, and therefore a few cases which have occurred in my practice of which I kept notes may be worthy of record.

In adults tetany is largely confined to women, being frequently associated with either pregnancy or lactation; it was primarily recognised by Trousseau in this connexion. It is only very occasionally—perhaps never—that it appears as a primary condition, being generally caused by some other disease of which it would seem to be a result. It has no special pathology of its own—at least, none has as yet been discovered—and it is apparently due to a reflex irritation of the excitable portion of the cortex—i.e., the motor areas, either alone or perhaps with the groups of cells in the anterior cornua of the cord. Clinically it is connected closely with epilepsy and chorea. Its association in adults with dilatation of the stomach is now established, and two of the cases which I have recorded below are an evidence of this point. The cases of this kind are mostly confined to the female sex. It may be assumed in these cases with gastric dilatation that it must be due to absorption from the stomach of toxins due to fermentation of food acting on the cells in the motor regions of the brain. In one of my cases this was manifest and the result was fatal. A similar explanation may be given of its occurrence in infants in association with diarrhœa and gastro-enteritis, in which conditions fermentation is nearly always present and toxins are in abundance, and even in rickets similar causes may be at work and may account not only for tetany but other nervous lesions, such as eclampsia and laryngismus, which are so frequent. Even those cases occurring in connexion with pregnancy or lactation, as well as those recorded by Weiss and Wölfer after excision of the thyroid gland, may possibly be explained by the same hypothesis—of some toxin present in the blood which affects the cortical grey matter. I have but six cases to record and four of these are in children.

CASE 1.—The patient was a male infant, aged seven months. He cut his first teeth at five months and was a healthy child. He had sudden attacks of convulsions—both tonic and clonic spasm—which continued for three days, when acute pneumonia appeared in the right lung. The convulsions continued intermittently for a week until the pneumonia began to clear up, when they ceased. This was followed by an attack of diarrhœa and sickness, which got better in a few days and left as a result constipation with large hard masses of fæces. The child was very much reduced by his illness and the tetany occurred while he was in this condition. There was marked tonic carpo-pedal spasm which was continuous even in sleep. The position of the hands was characteristic and the toes were flexed into the soles. There was considerable œdema of the face and of the backs of the hands and feet which were solid and non-pitting. There were jerky twitchings of the hands and arms. The condition lasted for some weeks and then got well. There was no evidence of rickets in this child. The treatment adopted was the administration of syrup of chloral and emulsion of cod liver oil. Carpo-pedal spasms are a rudimentary form of tetany, but are much more common than real typical tetany.

CASE 2.—The patient was a male infant, aged 15 months. There were no signs of rickets. He had been, and was at the time of treatment, a fine, healthy child. His first teeth had come when he was seven months old and he had got teeth rapidly within the following two months. He had been suckled and had lately had food as well. There was no history of diarrhœa or convulsions and no assignable cause for the present attack. There were contraction, tonic spasm, and œdema of both hands and feet. He had a cough with a crowing sound—"like a fowl crowing," his mother described it—evidently associated here with laryngismus which is essentially a rickety neurosis, still I could find no outward and visible signs of rickets.

CASE 3.—The patient was a female infant, aged three years, a stunted, rickety child, of about the size of a baby of eighteen months of age. I think this was the most marked case of rickets that I have ever seen. The child had never made any attempt to walk, she did not get a tooth until long after she was 12 months old, she had many attacks of both bronchitis and convulsions, and she had distorted joints and spine, &c. There was so much œdema of the face and hands that I examined the urine for albumin. The position of the hands and feet was just as in the other cases.

CASE 4.—This was an exactly similar case to the last. The patient was a stunted, rickety female child, aged two years, whom I had attended for both convulsions and bronchitis.

CASE 5.—The patient was a married woman, aged 41 years. The present was her third attack. The first had occurred 11 years before soon after her confinement, when she had got up and was suckling her infant. She had loss of feeling in the feet and pain and spasm in the hands and also, she said, loss of sight which lasted three days. She said that she felt at the time as if her face was greatly swollen but that it was not swollen and that this condition lasted then for six or seven weeks. She asserted also that she lost her speech for part of a day. Seven years ago she had another attack in just the same way after a confinement and while suckling her baby. This attack was not quite so severe as the previous one, but still it lasted for four or five weeks. She had recently had influenza. She had just nursed her daughter through a long attack of peritonitis, and she had recently moved from one house to another and had not got straight when the influenza occurred. I mention this to show that the patient had a good deal of both anxiety and worry. The position of the hands was characteristic and the spasm of the interossei so much that she could not separate the fingers, and my efforts to help her to do so evidently caused her great pain. There was a good deal of pain in the hands and arms. The feet were unaffected; sensation in both hands and feet was normal. On examining the abdomen I found marked dilatation of the stomach, reaching down to below the umbilicus. There were dulness at the lower level, a tympanitic note above, and a distinct splashing sound on succussion. Peristaltic action was evident and retention of food with periodical vomiting of large quantities of sour fluid. The condition disappeared in about a week. This case is interesting from the fact, if true, that she had temporary aphasia and loss of sight. It shows that the toxin which causes the tetanic spasm to appear is capable